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U.S. Department
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PESTS NOT KNOWN TO OCCUR IN THE UNITED STATES OR OF LIMITED
DISTRIBUTION NO. 77: PHILIPPINE DOWNY MILDEW

APHIS-PPQ

Prepared by L. W.H. Chang, Biological Assessment Support Staff,
PPQ, APHIS, USDA, Federal Building Room 634, Hyattsville, MD
20782

APHIS 81-49
September 1986

Disease

Philippine downy mildew

Pathogen

Peronosclerospora philippinensis (Weston) C. G. Shaw

Selected
Synonyms

Sclerospora philippinensis Weston
Sclerospora indica Butler

Class:
Order: Family

Oomycetes:
Peronosporales: Peronosporaceae

Economic
Importance

This pathogen causes a serious downy mildew disease to two major crops, corn and sugarcane. Yield losses of 40-60 percent in corn were common in some areas of the Philippines (Weston 1920). National yield loss for 1974-75 was estimated at 8 percent (Exconde 1976). No grain is produced because the seedlings quickly die or the plant slowly matures but produces little grain (Weston 1920). The percentage of yield loss correlates with the percentage of plants infected (Exconde and Raymundo 1974). In sugarcane, 45 percent infection resulted in about 25 percent loss of sugar per hectare in the Philippines (Husmillo 1982).

Hosts

Naturally infected hosts include Saccharum officinarum (sugarcane) (Husmillo 1982), Saccharum spontaneum (Chona and Suryanarayana 1955), Sorghum bicolor (sorghum) (Weston 1920), Tripsacum (Kenneth 1981), and Zea mays (corn, teosinte). Prevalence and severity in corn are much higher than in teosinte and sorghum (Weston 1920).

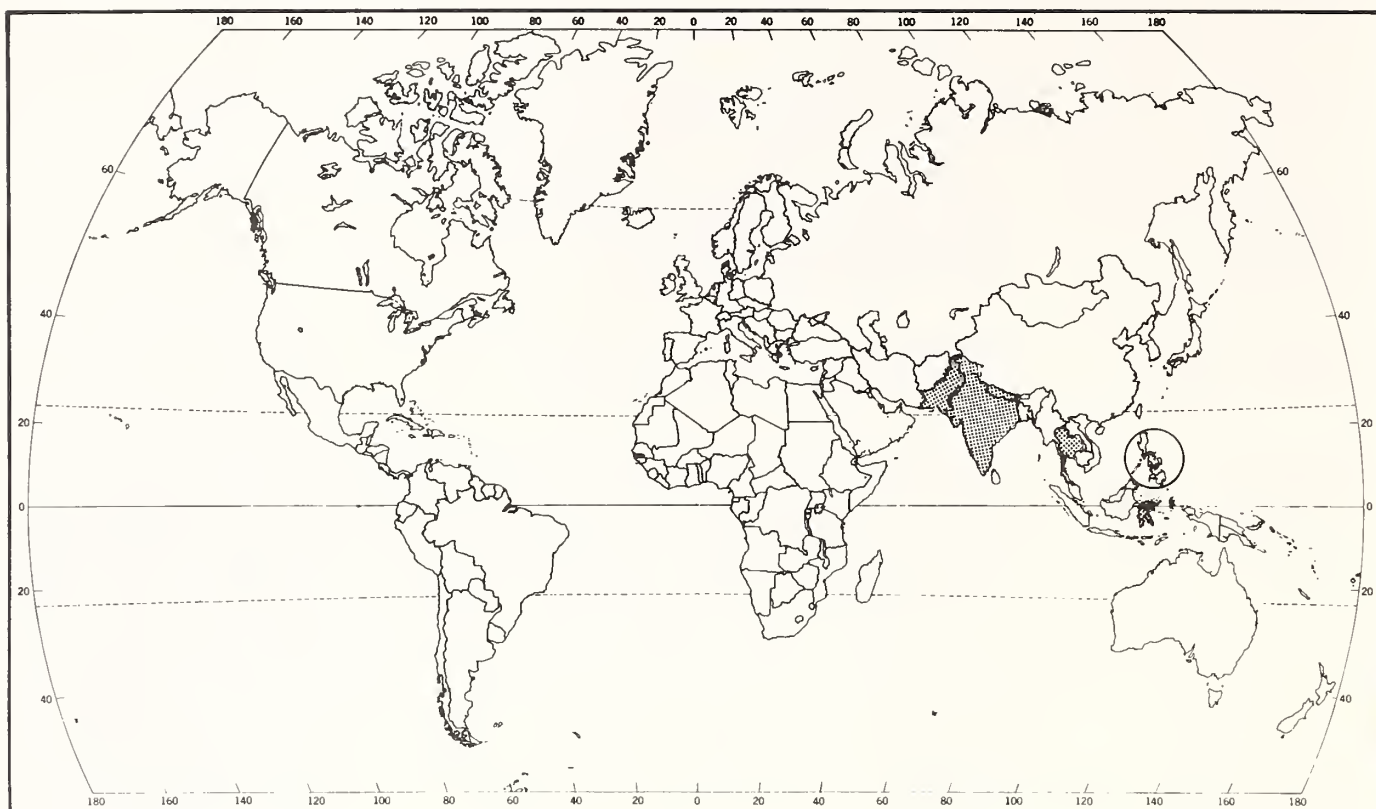
An inoculation study to determine the potential host range in the United States identified susceptible species from the grass tribes Andropogoneae (Andropogon, Bothriochloa, Eulalia, Saccharum, Schizachyrium, and Sorghum) and Maydeae (Tripsacum and Zea) (Bonde and Peterson 1983).

General
Distribution

India, Indonesia (only Celebes), Nepal, Pakistan, Philippines, and Thailand (Commonwealth Mycological Institute (1973, 1980)).

Characters

IMPERFECT STAGE - Conidiophore structure and dimensions, and spore shape and size greatly vary under different culture conditions and at different developmental stages. Mycelium intercellular in all parts of host except the root, branched, diameter 8 μ m, irregularly constricted and inflated; haustoria



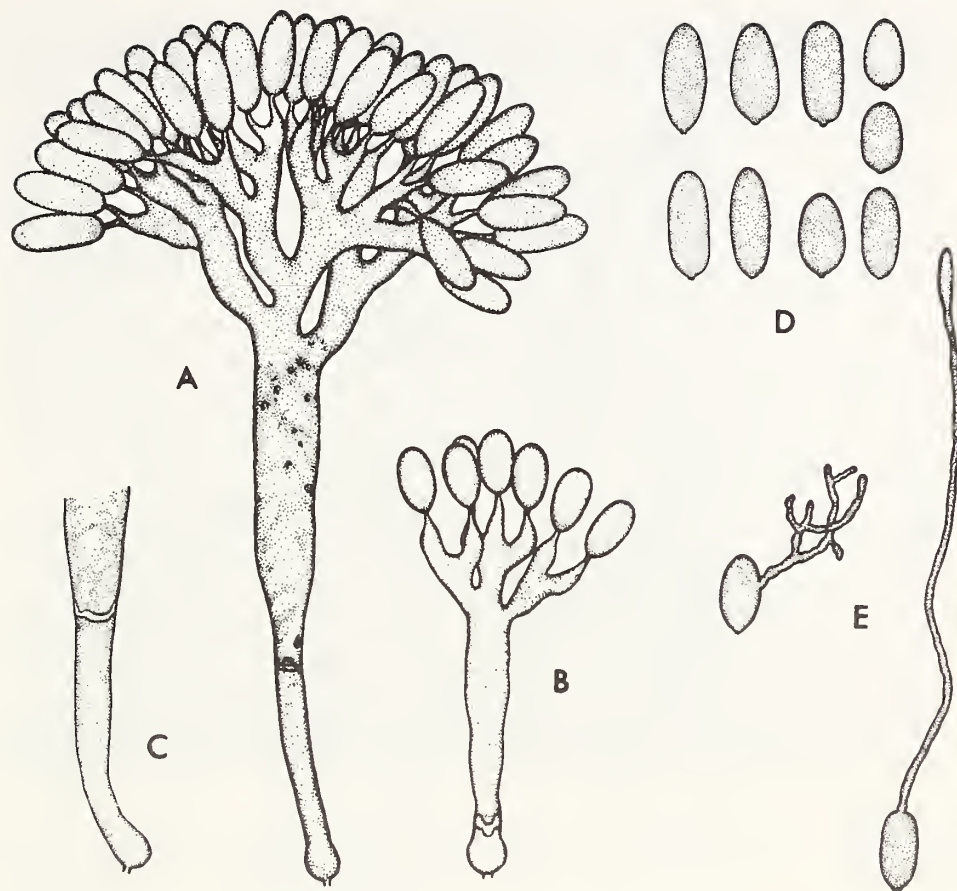
Peronosclerospora philippinensis distribution map (Prepared by Non-Regional Administrative Operations Office and Biological Assessment Support Staff, PPQ, APHIS, USDA.

simple, vesiculiform to subdigitate, $8 \times 2 \mu\text{m}$. Conidiophores (Fig. 1A-B) project through stomata, $150-400 \times 15-26 \mu\text{m}$; basal cell present (Fig. 1C); stalk cell dichotomously branched two to four times, branches robust, each branch may branch twice terminating in 2 or more sterigmata; sterigmata conoid to subulate, length $10 \mu\text{m}$, slightly curved (Weston 1920). Main axis widens abruptly base to apex (Visarathanonth and Exconde 1976).

Conidia hyaline; elongated ellipsoid, elongated ovoid, or rounded cylindrical (Fig. 1D); $27-39 \times 17-21$ (mostly 18×34) μm ; apex slightly rounded; base with minute apiculus; episporium thin; minutely granular within; germinate (Fig. 1E) by germ tube (Weston 1920).

PERFECT STAGE - None (Frederiksen and Renfro 1977).

(Fig. 1)



Peronosclerospora philippinensis. A. Conidiophore from corn during heavy dew. B. Stunted conidiophore from corn during light dew in hot, dry season. C. Basal cell of conidiophore. D. Conidia from corn. E. Germinated conidia. All 375 X (From Weston 1920).

Characters for this pathogen and for downy mildew pathogens that infect corn in the United States are compared in Table 1.

Characteristic Damage

Diseased corn generally exhibits chlorotic areas covered with downy mildew and deformed parts (Weston 1920). The size and outline of the streaks are defined on each leaf before it emerges (Dalmacio and Exconde 1969, Weston 1920). The downy mildew appears on the leaves and leaf sheaths and other affected parts except the roots. The fresh down is grayish white; matted fragments remain after drying by the sun. Deformities occur in various parts of the plant (Weston 1920). Symptoms are expressed at any time from seedling to silk stage.

Table 1. Comparison of Peronosclerospora philippinensis with domestic downy mildew pathogens on corn (Shurtleff 1980). Natural hosts as listed by Kenneth (1981).

	<u>Peronosclerospora</u>		<u>Sclerospora</u>	<u>Scleropthora</u>
	<u>philippinensis</u>	<u>sorghi</u>	<u>graminicola</u>	<u>macrospora</u>
Conidiophores	Hyaline, length 150-400 μ m, bloated, widening abruptly, dichotomously branched 2-4 times, ephemeral	Hyaline, length 180-300 μ m, bloated, often dichotomously branched 2-3 times, septate near base, ephemeral	Hyaline, length av. 268 μ m, bloated, nonseptate, irregularly dichotomously branched, ephemeral	Hyaline, length av. 13.8 μ m, simple, hyphoid, determinate
Asexual spores	Conidia hyaline, elongate-ovoid to round-cylindrical, apex slightly rounded, 27-39 X 17-21 μ m	Conidia hyaline, oval to almost spherical, 15-26.9 X 15-28.9 μ m	Sporangia hyaline, broadly elliptical, operculate, papillate, 14-23 X 11-17 μ m	Sporangia hyaline, lemon-shaped, operculate, 60-100 X 30-60 μ m
Germinate by	Germ tube	Germ tube	Zoospores	Many zoospores
Oospores	None	Usually brown to subhyaline spherical, diameter 25-42.9 μ m	Pale brown, spherical, usually smooth-walled, diameter 35 μ m	Hyaline to pale yellow, mainly in vascular bundles, diameter 45-75 μ m
Germinate by	Side germ tube	Wide germ tube	Germ tube	Sporangium
Hosts	<u>Saccharum</u> <u>Sorghum</u> <u>Tripsacum</u> <u>Zea mays</u>	<u>Panicum</u> <u>trypheron</u> <u>Sorghum</u> <u>Zea mays</u>	<u>Echinacloa</u> <u>Panicum</u> <u>Pennisetum</u> <u>americanum</u> <u>Setaria</u> <u>Zea mays</u>	<u>Avena sativa</u> <u>Echinacloa</u> <u>Eleusine</u> <u>Eragrostis</u> <u>Hordeum vulgare</u> <u>Iseilema</u> <u>Miscanthus</u> <u>Oryza sativa</u> <u>Paspalum</u> <u>Pennisetum</u> <u>Saccharum</u> <u>Setaria</u> <u>Sorghum</u> <u>Triticum</u> <u>aestivum</u> <u>Zea mays</u> , etc.

(Fig. 2)



Philippine downy mildew in very young corn plant showing dwarfing, overall paleness, narrow and stiff leaves, and narrow striping of later leaves along their length (From Weston 1920).

The intensity of the symptoms is governed by the age of the plant when infected which controls its susceptibility, modified by the resistance of the cultivar and other factors. Plants 4 weeks old or older are almost immune. These plants exhibit a local chlorosis: pale green, elongate streaks or minute dots, the latter in late infections of older plants. Seedlings less than 4 weeks old are susceptible and display the systemic symptoms described below (Dalmacio and Exconde 1969, Weston 1920).

Infected very young, seedlings (two to four leaves nearly developed and four to seven partly developed) show severe symptoms (Fig. 2). The first symptom appears at the base of one of the first to fourth leaves as two or three narrow, long, pale yellow to whitish stripes; succeeding leaves are almost completely whitish or pale yellow. Diseased leaves are narrow and ascend stiffly instead of bending. The plant is stunted with leaf sheaths often overlapping and enclosing the tassel. Roots are usually stunted. The plant may quickly die or slowly mature and produce an ear, sometimes with a few kernels (Weston 1920).



Philippine downy mildew in older corn. Left - healthy plant. Right two - diseased (From Weston 1920).

Infected somewhat later, plants exhibit the first symptom on one of the fourth to eighth leaves (Fig. 3). The base of the leaf exhibits pale stripes larger than that in early infected plants. Striping intensifies on succeeding leaves with stripes on each, running closer to the leaf tip. The stripes usually merge at the base of the lower leaves into a yellowish white area, the middle leaves show a smaller blotch, and on the upper leaves, broken stripes merge laterally to present a mottled appearance. Some midribs may break where they join the sheath, leaving the leaf hanging straight down the stalk. Reproductive structures are also affected. Ear production is retarded. Tassels and ears may show a wide range of malformations. Less pollen and few kernels, if any, are produced. As the diseased

plant matures and sporulation almost stops, chlorotic areas become greener until in plants that are less severely affected, recovered areas resemble healthy areas (Weston 1920).

If tillers become infected on a healthy main plant, infection can spread into the main stalk. The lower leaves show interrupted, narrow, pale, yellow-green to rusty-green stripes along their length; middle leaves are so marked in the more distal end; upper leaves are entirely unmarked or marked in the leaf tip. No conidiophores emerge (Weston 1920).

Diseased sugarcane leaves at first exhibit reddish-brown specks that elongate into short streaks along the veins. Succeeding leaves show increasingly pale yellow to whitish discoloration from base to tip (Elazegui and Exconde 1968).

Detection Notes

Movement of infected plant parts of the hosts of P. philippinensis could introduce it into new areas. To prevent the introduction of exotic pathogens on corn, sorghum, sugarcane, and other grass relatives into the United States, Title 7 of the Code of Federal Regulations regulates the entry of these commodities under Parts 319.15, 319.24, 319.37, and 319.41.

Survey for diseased plants by examining corn seedlings that have larger and larger yellow streaks on succeeding leaves. Grayish white down will be in the chlorotic areas although sun or low humidity may have dried the mildew to matted fragments. Older plants may exhibit sterile tassels or ears. Other plants may be stunted or dead. Sugarcane has some of these symptoms.

Submit specimens for identification by packing diseased material in double containers (one container inside another) with screw lids.

Biology and Etiology

Perennial grass hosts serve as reservoir hosts to carry over the pathogen during unfavorable periods, or provide primary inoculum (Bonde 1982). The known infective agents are the mycelium in these plants or the airborne conidia produced by the mycelium. Seedborne transmission may occur in corn.

In the host, the mycelium produces the conidiophores, which bear the conidia. The conidiophores emerge through host stomata in the chlorotic areas on both leaf surfaces (more so on the lower surface of corn because of more stomata), and on sheaths, tassel rachis, glumes, and husks (Dalmacio and Exconde 1969). Conidial production, germination, and infection require nights of 21-26° C and a thin film of moisture, such as dew (Dalmacio and Raymundo 1972). Moisture for 4-5 hours in darkness is

essential for spore formation (Bonde 1982, Exconde 1970). Conidia form sparsely the morning after the first symptom appears, and abundantly after systemic symptoms appear (Dalmacio and Exconde 1969). Sporulation may begin at midnight and continue until surface moisture dries (Weston 1920). Sporulation on individual plants can continue for more than 2 months, releasing enormous numbers of conidia (Bonde 1982). Wind currents and splashing water disperse the conidia.

After the conidia land, 2 hours of free moisture on the host surface is required for germination and penetration (Bonde 1982). Germination is optimum at 19-20° C (Exconde 1970) or 15-33° C (Bonde 1982), but readily continues at 6.5° C. Once exposed, however, to drying conditions (such as sun, wind, or low humidity for 1-2 hours) the conidia shrivel and no longer germinate (Weston 1920). Viable conidia germinate in less than 1 hour under favorable conditions. Penetration of the host occurs about 2 hours after inoculation (Dalmacio and Exconde 1969).

Conidial germ tubes or hyphae from the germ tube penetrate corn leaves through the stomata and spread intercellularly through the mesophyll cells. The fungus grows mainly downward through the leaf sheath to the stem where it moves into and persists in the shoot apex. Williams (1984) suggests that the susceptibility of very young plants and resistance of older ones may be accounted for by the ability of the fungus to penetrate the growing point of a shoot before, but not after, the point fully differentiates. When the mycelium does invade the meristematic tissue, chlorotic streaks soon appear on the leaves, followed by the fungus sporulating in these areas when conditions are favorable, producing the secondary inoculum (Dalmacio and Exconde 1969).

The fungus spreads throughout the plant, but it is confined to the chlorotic, not the green, areas. Besides the leaf blades and sheaths, it also invades tassels, glumes, and ears (Dalmacio and Exconde 1969). Weston (1920) described the hyphae as "intercellular in all parts except the root," but stated elsewhere in his paper that the root "is not extensively invaded." Dalmacio and Exconde (1969), however, observed mycelium in the root, mainly in the cortex.

In the laboratory, corn seed from these plants produce infected seedlings, especially when hard dough kernels are planted soon after harvest. Because infected plants mature slower than uninfected plants, ears from the former are left in the field while the latter are harvested. The infected ears may then become a new inoculum source. No infections result from inoculations of healthy seed with conidia. The infective agent

is the mycelium within the infected seed, reported located in the pericarp (Advincula and Exconde (1976) or the embryo (Bains and Jhooty 1982).

The number of systemically infected (nonyielding) plants is positively correlated with night relative humidity, spore production, day relative humidity, and rainfall; and negatively correlated with night and day temperatures and duration of sunlight (Bonde 1982, Exconde 1976).

Cultural Controls

Resistant cultivars appear to be the best means of control. Sanitation and roguing are also practiced. Protection of young plants during the most susceptible period helps prevent infection (Holliday 1980). Drying infected corn seed to 14-30 percent moisture destroys the fungus and prevents seedborne transmission (Advincula and Exconde 1976).

Literature Cited

Advincula, B. A.; Exconde, O. R. Seed transmission of Sclerospora philippinensis Weston in maize. Philipp. Agric. 59:244-245; 1976.

Bains, S. S.; Jhooty, J. S. Distribution, spread and perpetuation of Peronosclerospora philippinensis in Punjab (India). Indian Phytopathol. 35(4):566-570; 1982.

Bonde, M. R. Epidemiology of downy mildew diseases of maize, sorghum, and pearl millet. Trop. Pest Manage. 28(1):49-60; 1982.

Bonde, M. R.; Peterson, G. L. Comparison of host ranges of Peronosclerospora philippinensis and Peronosclerospora sacchari (downy mildew, Philippine and Taiwanese strains). Phytopathology 73(6):875-878; 1983.

Chona, B. L.; Suryanarayana, D. The occurrence of Sclerospora philippinensis Weston on 'kans grass' (Saccharum spontaneum L.) in India. Indian Phytopathol. 8:209-210; 1955. Taken from Rev. Appl. Mycol. 36(4):241-242; 1957.

Commonwealth Mycological Institute. Pathogen: Sclerospora philippinensis Weston. London: Commonwealth Agricultural Bureaux; 1973. (Distribution maps of plant diseases, no. 497, ed. 1). Additions and corrections; 1980.

Dalmacio, S. C.; Exconde, O. R. Penetration and infection of Sclerospora philippinensis on corn. Philipp. Agric. 53(1):35-52; 1969.

- Dalmacio, S. C.; Raymundo, A. D. Spore density of Sclerospora philippinensis in relation to field temperature, relative humidity and downy mildew incidence. Philipp. Phytopathol. 8(1/2):72-77; 1972.
- Elazegui, F. A.; Exconde, O. R. The basis of differentiating Sclerospora philippinensis Weston as a species distinct from Sclerospora sacchari Miyake. Philipp. Agric. 51(10):767-778; 1968.
- Exconde, O. R. Philippine corn downy mildew. Indian Phytopathol. 23(2):275-284; 1970.
- _____. Philippine corn downy mildew: Assessment of present knowledge and future research needs. Kasetart J. 10(2):94-100; 1976.
- Exconde, O. R.; Raymundo, A. D. Yield loss caused by Philippine corn downy mildew. Philipp. Agric. 58(3/4):115-120; 1974.
- Frederiksen, R. A.; Renfro, B. L. Global status of maize downy mildew. Ann. Rev. Phytopathol. 15:249-275; 1977.
- Holliday, P. Fungus diseases of tropical crops. Cambridge, England: University Press; 1980: 453-454; 1980.
- Husmillo, F. R. Assessment of yield loss due to downy mildew of sugarcane caused by Peronosclerospora philippinensis (Weston) C. G. Shaw. Sugarcane Pathol. Newsl. 28:17-24; 1982.
- Kenneth, R. G. Downy mildews of graminaceous crops. Spencer, D. M., editor. The downy mildews. New York: Academic Press; 1981:367-394.
- Shurtleff, M. C., editor. Compendium of corn diseases. 2d ed. St. Paul, MN: American Phytopathological Society; 1980: 29, 34-35.
- Visarathanonth, N.; Exconde, O. R. Development, morphology and cytology of sporulation in Philippine Sclerosporas. Philipp. Agric. 59(7&8):266-287; 1976.
- Weston, W. H., Jr. Philippine downy mildew of maize. J. Agric. Res. 19(3):97-122; 1920.